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Case report

Fatal pulmonary hemorrhage after taking anticoagulation medication

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ABSTRACT

We describe a 64-year-old man with extensive diffuse acute lung hemorrhage, presumably as a result of anticoagulation therapy. We evaluated reports in the literature concerning acute exacerbation (acute lung injury of unknown cause) in UIP and other forms of fibrotic interstitial pneumonias. We also evaluated autopsy tissue in this case in order to determine the cause of death in this 64-year-old man, who was initially thought to have an asbestos-related disease. Based on the autopsy findings, this man died as a result of anticoagulation therapy; specifically, the use of Xarelto® (rivaroxaban).

1. Case report

A 64-year-old Caucasian male underwent right total knee replacement on December 4, 2013 and was subsequently prescribed a 21-day supply of the anticoagulation drug Xarelto® (rivaroxaban), 10 mg by mouth every day with dinner. Approximately two weeks had elapsed since he stopped taking Xarelto when, on January 13, 2014, the patient was admitted to the hospital with a violent, nonproductive cough and extreme shortness of breath. He denied fever. He had an elevated white blood cell count of 20,400 K/uL. CT scan showed ground-glass opacity and bilateral atypical pneumonia. He was treated with antibiotics (azithromycin 500 mg daily and ceftriaxone 100 mL NS, 1 g q24h). He was started on subcutaneous heparin (enoxaparin 40 mg/0.4 mL) at bedtime between 1/14/14 and 1/26/14 for prevention of deep venous thrombosis (DVT)/pulmonary embolism (PE) for a total of 12 doses.

Other medical problems included pulmonary nodules; prostatic hyperplasia; obstructive sleep apnea; hyperlipidemia; left shoulder rotator cuff repair; and recent knee replacement surgery. He had a prior history of asbestos exposure and there was concern he might have an asbestos-related disease.

A chest radiograph and CT scan showed stable cardiomegaly, a right pneumothorax, diffuse interstitial thickening with patchy airspace opacities, and lack of significant honeycombing. The abnormalities were stated to have significantly progressed since September 2011.

The patient was thought to have community acquired pneumonia on top of pre-existing asbestosis and interstitial lung disease. Video-assisted thoracoscopic lung biopsy was performed on January 21, 2014. Preoperative and post-operative diagnoses were: 1) probable interstitial lung disease; 2) asbestosis; and 3) atrial arrhythmia. Operative findings stated the surface of the right lung was abnormal. Wedge biopsies of the right middle and lower lobes were obtained and pathologic evaluation revealed patchy interstitial fibrosis with chronic inflammation consistent with usual interstitial pneumonia (UIP), with focal organizing pneumonia and progression toward end-stage lung.

A progress note dated January 23, 2014 stated the patient was to remain on ceftriaxone for 14 days; IV steroids for interstitial lung disease; metoprolol for atrial fibrillation; Prevacid for gastrointestinal (GI) prophylaxis; Dilaudid for pain control; enoxaparin (heparin) for DVT prophylaxis; and N-acetyl cysteine (NAC) 600 mg BID.

The patient started coughing up bloody mucus on 1/23/14. His chest tube was removed on post-op day #4 and he was restarted on 20 mg of Xarelto every evening between 1/26/14 and time of discharge on 2/7/14. He was also started on IV Lasix without improvement so his Lasix was increased to 40 mg BID IV. Upon discharge from the hospital, he was switched to oral furosemide (Lasix) 40 mg daily for 30 days.

The patient had a prolonged hospital stay (26 days) and was discharged home to hospice care with a prognosis of two weeks to one year. He had severe dyspnea on minimal exertion. In fact, in the course of one month, he progressed from being ambulatory and independent to being oxygen-dependent and requiring complete care. He continued his prescription of 20 mg of Xarelto at home.

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Discharge medications included:

1. Prednisone 30 mg b.i.d.
2. Benzonatate 200 mg every 8 h PRN.
3. Diltiazem 240 mg every day.
4. Furosemide (Lasix) 40 mg daily.
5. Prevacid 30 mg every day.
6. Toprol-XL 25 mg every day.
7. Morphine sublingual 5–10 mg every 2 h PRN with 30 mL dispensed.
8. Ativan 0.5–1 mg every 8 h PRN with #30 dispensed.
9. Rivaroxaban (Xarelto®) 20 mg daily [at home].
10. Metformin, 500 mg twice daily.

Discharge diagnoses were:

1. Idiopathic pulmonary fibrosis, status-post open lung biopsy.
2. Asbestosis [note: this as a clinical diagnosis. While he had a history of asbestos exposure, he did not have radiographic or pathologic asbestosis].
3. Acute-on-chronic hypoxemic respiratory failure.
4. Atrial fibrillation with rapid ventricular response.
5. Steroid-induced hyperglycemia treated with metformin.
6. Lower extremity edema from cor pulmonale and steroids, treated with Lasix.

The patient's condition continued to deteriorate and he expired on February 15, 2014. An autopsy was performed on February 19, 2014.

1.1. Autopsy findings

The decedent measured approximately 71″ to 72″ tall and weighed an estimated 250 pounds. There was mild rigor and livor mortis. Examination of the thorax revealed a scar at approximately the 10th or 11th rib on the right. Ribs and sternum were normal. The mediastinal tissue was normal. No hyaline pleural plaques were identified. The pleural cavities did not contain fluid. The trachea was normal.

The right lung, fixed, weighed 1231.5 g and measured approximately 22 × 16 × 12 cm. The surface of the right lung was hemorrhagic with petechiae and was somewhat nodular. The visceral pleural surface was finely nodular. Upon sectioning, the parenchyma was extremely hemorrhagic. There were a few nodules on the pleural surface. No thrombi or thromboemboli were identified. There were no hyaline pleural plaques on the visceral or parietal pleura, including the surface of the lungs.

The superior vena cava was normal. The heart was in its usual anatomic position and appeared normal. No tumor was identified on the pericardium. There was blood on the parietal pleural surface. There did not appear to be blood in the trachea or bronchi.

The left lung, fixed, weighed 1038 g and measured approximately 22 × 15 × 8 cm. There was nodularity on the visceral pleural surface. The surface of the left lung was hemorrhagic with petechiae. Upon sectioning, the parenchyma was extensively hemorrhagic.

The macroscopic appearance of the lungs is shown in Figs. 1 and 2.

Microscopically, the lungs showed extensive hemorrhage (Fig. 3) with filling of alveolar spaces with fresh blood. There were areas of hyaline membrane formation (Fig. 4) with bright red material lining the alveolar spaces. There was subpleural interstitial fibrosis with some honeycombing, the findings being consistent with usual interstitial pneumonia (Fig. 5). There was mild interstitial fibrosis. There were aggregates of macrophages in alveolar spaces. No neoplasm was identified. Trichrome stained sections showed peripheral interstitial fibrosis and blood (Fig. 6). Iron stains showed hemosiderin (Figs. 7 and 8).

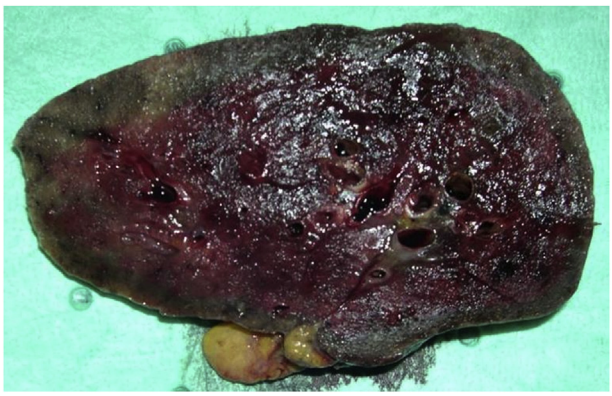

Fig. 1. Macroscopic appearance of lung.

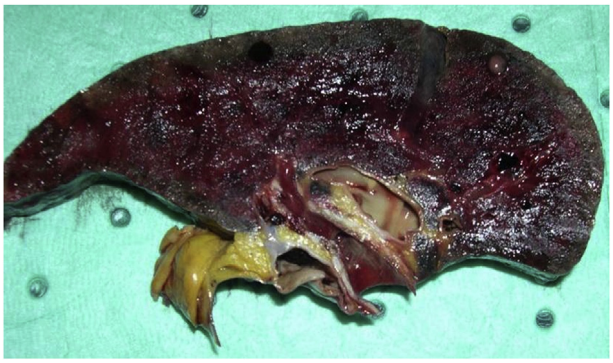

Fig. 2. Macroscopic appearance of lung.

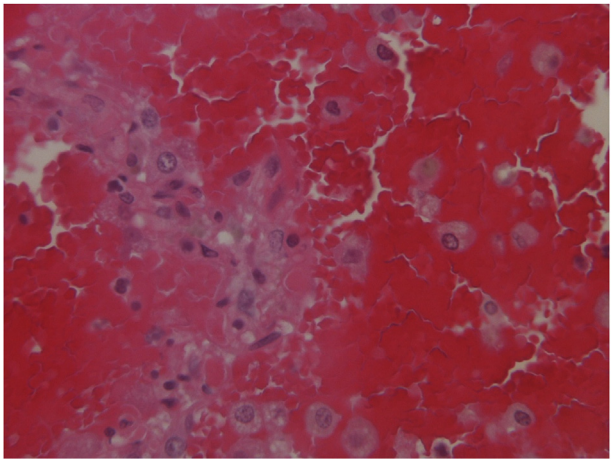

Fig. 3. Extensive intrapulmonary hemorrhage.

2. Acute lung injury of unknown cause

We evaluated reports in the literature concerning acute exacerbation (acute lung injury of unknown cause) in UIP and other forms of fibrotic interstitial pneumonias [1–4]. These reports of acute lung injury did not provide any mechanism by which these acute processes developed. In the report by Swigris et al. [2], the